P S E U D O E C L A M P S I A

with

RECORD OF CASES.

THESIS for the Degree of Doctor of Medicine

by

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PSEUDO ECLAMPSIA.

In the life work of an Obstetrician there is perhaps no moment more fraught with anxiety than that in which he finds a pregnant, parturient, or puerperal woman, in the throes of a convulsion. Instinctively his diagnosis is Eclampsia, and the high mortality rate of the disease flashes before his mental vision.

But after all, Eclampsia may not be the true diagnosis. Convulsions per se do not necessarily mean Eclampsia. Other diseases of which convulsions are, or may be, symptoms, occur in pregnant, parturient, and puerperal patients; and the object of my thesis is to study such diseases to which I venture to apply the name of Pseudo Eclampsia.

Eclampsia usually occurs in primiparous women. Of 306 cases Schauta found 253 were in primiparae, and Chautreuil states that in 683 cases only 161 were multiparae.

Etiology - The causal agent is a Toxin, the unvarying expression of which is the presence of albumen in the urine.

Symptoms.

The woman is found suffering from a convulsion. On enquiry it is found that for some days she has not been in her usual health. She may have had headache, some/

some visual disturbances, either dimness of vision or muscae volitantes.

There may have been gastric symptoms either epigastric pain or vomiting.

It may have been noted that her face seemed swollen and that it had a 'pasty' look, the puffiness being especially noticeable under the eyes. There was some oedema of the limbs - the impress of her stockings being observed over her ankles at night.

There had been marked constipation.

A very important feature is the history of diminution of the amount of urine passed daily. The urine is high coloured and examination of it would reveal the presence of a large percentage of albumen.

The above are premonitory signs of Eclampsia and they are allowed to exist practically unheeded by the patient, though to the practitioner they would indicate that his patient is bordering on one of the most serious illnesses to which a woman can be subjected.

These signs being ignored the storm bursts and the woman has a convulsion which has three definite phases.

(a) The Tonic phase.

(b) The Clonic phase.

(c) The Relaxation phase with Coma.

Time of onset of convulsions.

The/

The convulsion may occur before, during, or after labour - but it has been found that their occurrence is most common during the first stage of labour. Schauta found that convulsions began during labour in 185 cases; that they preceeded labour in 42 cases, and that they followed labour in 82 cases.

When the convulsions precede labour they usually occur in the last two months of pregnancy.

The convulsion.

The attack is sudden in its onset, and in many of its characteristics is precisely similar to that of a severe Epileptic convulsion. Close observation will show that there is at first a short period of tonic spasm affecting the entire muscular system, voluntary, as well as involuntary. This is succeeded almost immediately by violent clonic contractions generally commencing in the muscles of the face which twitch violently; the expression is horribly altered; the eyeballs are turned up under the eyelids so as to leave only the white sclerotics visible. The angles of the mouth are retracted and fixed in a convulsive grin. The tongue is at the same time forcibly protruded, and, if care be not taken, it is apt to be lacerated by the violent grinding of the teeth. The face at first pale soon becomes livid and cyanosed, while/

while the veins of the neck are distended, and the carotids are seen to pulsate vigorously. Frothy saliva collects about the mouth and the whole appearance is so changed as to render the patient unrecognisable.

The convulsive movements soon attack the muscles of the body. The hands and arms, at first rigidly fixed with the thumbs clenched into the palms, begin to jerk, and the whole muscular system is thrown into rapidly recurring convulsive spasms. This is shown by a temporary arrest of the respiration at the commencement of the attack, followed by irregular and hurried respiratory movements which cause a peculiar hissing sound.

During the attack the patient is absolutely unconscious; sensibility is totally suspended and she has afterwards no recollection of what has taken place. Fortunately the convulsion is not of long duration, and, at the outside, does not last more than three or four minutes; generally not so long. In most cases after an interval there is a recurrence of the convulsions characterised by the same phenomena, and the paroxysms are repeated with more or less force and frequency according to the severity of the attack.

The convulsion is never followed by an immediate return to consciousness but always by coma which is

of/

of varying duration and of varying depths, gradually passing into stupor during which the patient may indicate that she is suffering severe pain in her head.

The urine is greatly diminished in quantity and the catheter may be able to evacuate only a few ounces from the bladder. The resulting specimen has a typical smoky appearance. On examination the specific gravity is found to be lowered - 1010 being an average figure - as compared with 1020 - the specific gravity of the urine of a healthy individual. The test for albumen reveals that substance in great quantity, in fact when a small quantity of urine is boiled it becomes quite solid in the test tube. Blood is invariably present.

On microscopical examination tube casts of all varieties - blood, granular, and fatty - are found. The total amount of urea is greatly diminished.

The blood pressure is found to be considerably heightened, 170 to 200 m.m. of Hg. being an average reading as compared with 120 m.m. of Hg. which is regarded as the Sphygmomanometric reading of a normal individual.

The temperature is usually slightly raised - up to 100 F - but it fluctuates from hour to hour; and after each convulsion it tends to rise higher.

To recapitulate, therefore, the chief points in

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a typical case of Eclampsia are:-

1. The premonitory Signs.
2. The sudden onset of Convulsions.
3. Urinary Disturbances.
    (a) Diminution in amount.
    (b) Lowering of Specific Gravity.
    (c) Presence of large percentage of albumen.
    (d) Presence of blood.
    (e) Diminution of Urea output.
    (f) Presence of Urinary Casts.
4. Great rise of the blood pressure.

Two factors of great importance constitute Eclampsia, one, convulsions, the other albuminuria; and it is my contention that when there are convulsions in a pregnant, parturient, or puerperal woman without albuminuria, that patient is not suffering from Eclampsia but from a condition which comes under the category of Pseudo Eclampsia.

I propose to record three cases - excellent examples of Pseudo Eclampsia - which occurred at the Royal Maternity Hospital of Edinburgh during the Quarter, October, November, and December, 1912 - the service of Sir Halliday Croom - and during my term of house surgeonship there.

These cases were all sent in to Hospital with the diagnosis of Eclampsia. The first case was only seven and a half months pregnant; the second was actually in labour; whilst the third was a puerperal case. They are, therefore, cases resembling Eclampsia at the various times during which it can occur. The first case eventually turned out to be a case of Cerebral Tumour; the second a case of Hysteria; and the third a case of Tuberculous Meningitis.

CASES OF CEREBRAL TUMOUR.

Case I

Name. H. M.

Aged 24½.

1 para. 7½ months pregnant illegitimately.

Occupation. Potato Digger.

Admitted to Royal Maternity Hospital, Edinburgh Monday, October 28th, 1912.

History.

Patient two days before admission was in Edinburgh, and at one of the Railway Stations was seized with what her friends recognised to be a convulsion. She did not recover quickly; she was therefore taken in a cab to her house some eight miles distant. She was put to bed but continued taking convulsions all night. Her friends estimate that the attacks recurred each ten minutes.

On the following day, October 27th, a doctor was summoned and recommended the patient's removal to Hospital. His advice, however, was unheeded, and patient continued having convulsions all the day. Next day, October 28th, the Doctor was again summoned and he now insisted on patient being removed to Hospital, where she was admitted at 12-15 p.m.

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A trained nurse accompanied her in a cab and stated that during the journey - which occupied one hour and a quarter - she (the patient) had six convulsions.

She had always been a healthy woman, and had been working as a potato digger up till the day of her illness. She had been confined four days previously without untoward symptoms.

State on Admission.

Patient is a healthy looking, well built, well nourished woman, aged 24½. Her face has a 'bright and brilliant' hue, just as if she has scarlet fever - but her temperature registers 98·4 F. and her pulse rate is 88 beats per minute.

Os admits one finger. Multiparous os.

Blood pressure 140 m.m. of Hg.

Circulatory System - nothing abnormal to be made out. Valves closed at all areas.

Respiratory System appears quite normal.

Patient is comatose.

A catheter was passed and nine ounces of urine were drawn off.

Characters:-

Clear.

No albumen.

No blood.

No Bile.

No sugar

No Acetone.

No Casts.

The Specific Gravity was not estimated as the quantity of urine obtainable was very small. The pupils were equal, moderately dilated and reacted equally to light.

Reflexes.

Knee jerks - on both sides very active - equally so.

No ankle clonus.

Extensor response of great toe of each foot, more active on the left foot than on the right.

Progress and Treatment.

Twenty minutes after admission patient had a convulsion. It was slight. There were present the typical facial twitchings and the jerkings of the limbs. These movements began in the face, and extended to the limbs - both sides of the body being equally affected. She did not become purple, had not the markedly congested look, and the stertorous breathing usual at the end of such a seizure was conspicious by its absence.

There was given a large enema, and later the colon was washed out. The results were disappointing - only a few scybalous masses being returned. The stomach

was/

was washed out and a copious lavage resulted - much fluid and solid being returned. It smelt strongly of beer. Lavage was continued till the return was quite clear and then there was left in the stomach six ounces of Magnesium Sulphate dissolved in as little water as possible. This was retained.

From admission at 12-15 p.m. till 3 p.m. patient had seven convulsions - none very severe and each having the characteristics already described.

At 3 p.m. she was given ¼ grain Morphia Sulphate hypodermically.

The blood pressure was still at 140 m.m. of Hg.

She was put between blankets and surrounded with hot water bottles.

At 4-30 p.m. Sir Halliday Croom saw the patient, and said the case not one of ordinary Eclampsia; at this time the patient had more and more the look of a person ill with Scarlet Fever.

While she was being vaginally examined at 5 p.m. she took a very severe convulsion. She became at this time livid and cyanosed, and sterterous breathing was very evident. So severe was the attack that ether had to be administered. This was the first convulsion since 3 p.m. - when the morphia was given - and it brought the total up to eight. It was by far the

most/

most severe convulsion the patient had since admission to Hospital.

At 6 p.m. she was venesected and six ounces of blood withdrawn. The blood pressure fell to 100 m.m. of Hg. She required no anaesthetic, showed no signs of a convulsive attack despite this surgical interference - in fact she never moved a muscle during the whole procedure.

Up till 8 p.m. she had no further convulsions, but remained in a very restful condition.

After 8 p.m. she was much more easily roused. By speaking in just a moderate tone one easily attracted her attention. She opened her eyes but made no attempt whatever to speak. She was able, however, to swallow some fluid. From 8 p.m. till 1 a.m. (October 29th) she had five "feeders" of water - in all about thirty ounces.

At 2 a.m. October 29th patient's breathing became very quick and she was spitting a good deal of frothy fluid. This was causing her such discomfort that the stomach tube was again passed and the stomach emptied.

The bowels still refused to act.

10 a.m. October 29th.

Patient had no further convulsions. Blood pressure 92 m.m. of Hg. Temperature 103^{0} F, and pulse 124 per minute. Knee jerks much less active but present.

Extensor/

Extensor response obtainable only from left great toe - the response from the right great toe is now a flexor one. No ankle clonus.

As bowels still refused to act patient was given Croton Oil minims III on butter. This she was unable to swallow. It stuck at the back of her throat and caused such great embarrassment of respiration that it had to be swabbed away. She retched a good deal and brought up some bile stained fluid. Her stomach, accordingly, was again washed out with a solution of Bicarbonate of Soda and much bile coloured fluid removed.

She was given another enema - but still without result. As the tube did not pass easily, the rectum, lest any faecal mass were blocking its passage, was digitally examined. No such obstacle could be felt.

Patient's temperature rose from 103^{0}F to 107^{0}F; she gradually sank; and died at 5-45 p.m., October 29th, 1912.

The urine on October 29th contained just the merest trace of albumen - probably due to pyrexia - and during all that day she passed urine copiously in bed.

A post mortem examination was made on October 30th, 1912, and the following report furnished:-

"Body well developed and well nourished. Rigidity well/

well marked. Post Mortem changes along line of vessels. Lividity of face and neck.

Pleural Sacs empty - no adhesions.

Pericardial Sac contains slight excess of fluid.

Peritoneal Sac contains slight excess of fluid.

Lungs show oedema and in lowest portions - congestion.

Heart - Cavities contain post mortem clot in large amount. Valves normal. Muscle pale, soft, and friable. Diffuse fatty degeneration.

Liver - Well Marked fatty degeneration. No haemorrhages.

Spleen - Slightly enlarged and congested.

Kidneys - Left - Small soft pale well marked fatty and catarrhal changes. Vessels congested.

Right - Larger- but shows similar pathological changes.

Stomach and Intestines - uniformly distended with gas, no abnormality.

Brain -

Vessels greatly congested. In left anterior parietal region towards the inner surface is a lobulated firm tumour - well defined from the surrounding brain. It is about the size of a tangerine orange.

No abnormality of the Pituitary Body.

Uterus greatly enlarged and contains a foetus. Veins distended with post mortem clot.

Summary:-/

Summary:-

Large tumour of left anterior parietal Region.
Fatty and Catarrhal change in Kidneys.
Fatty Degeneration of Liver and of Heart.
Oedema of Lungs"

The tumour on microscopical examination proved to be a Cholesteatoma - or "Pearl Tumour" - usually associated with, and growing from the meninges.

Beattie and Dickson say "This tumour is excessively rare in the substance of the brain. It is composed mainly of laminated layers of squamous epithelial cells and usually a central mass of Cholesterin crystals. It is supposed to originate from the Epithelium of the ventricles.

Ziegler mentions the presence of small hairs in these tumours and regards them as Dermoids.

The Exact Localization of the Tumour.

The tumour reaches to within half an inch of the anterior end of the left hemisphere and lies opposite the Superior Middle and Inferior Frontal Convolutions of the convex surface of the hemisphere; and opposite those parts of the Marginal and Callosal Convolutions (on the inner surface) which lie above the anterior half of the Corpus Callosum. Where it lies in relation to the Superior Frontal Convolution it is near the/

the surface, and the fibres arising from and ending in that convolution must have suffered great displacement and compression. It is about ½ inch away from the surface of the Middle Frontal convolution and one inch from the inner surface of the inferior, and fibres from these have therefore suffered less although they would have been less affected. On the inner aspect of the hemisphere where the tumour is at the surface the Marginal and Callosal convolutions have been well nigh obliterated in the region involved only shreds of cerebral tissue remaining on the surface of the tumour, and in that region there is no evidence of the remains of the Cingulum - the long association tract associated with the Callosal convolution. In this region the tumour has grown beyond the plane of the surface of the left hemisphere and has produced a depression on the corresponding part of the Marginal and Callosal convolutions of the right hemisphere, probably sufficient to have produced manifestations of compression if that part of the brain had had functions which control the outward acts and appearances. The tumour has passed the anterior part of the left half of the Corpus Callosum backwards and downwards, stretching it and thinning it out and through it exerting pressure on the head of the Caudate Nucleus - which has been compressed and flattened from above downwards and/

and at one point the tumour has burst through the Corpus Callosum invaded the head of the Caudate Nucleus and crushed the anterior limb of the internal Capsule. The posterior limit of the tumour is at a level one inch in front of the posterior end of the Corpus Callosum and at its lowest level it is about half an inch above the surface of the posterior part of the anterior Orbital convolution.

The middle frontal convolution has been said to contain motor centres for the orbital muscles of the right side. The Callosal convolutions may have to do with smell; the rest of the regions involved have not yet been ascribed definite functions but it has been supposed that in these are evolved those higher processes of thought of which the patient's occupation did not provide sufficient opportunity for adequate expression.

This case presents many interesting features. That such a large tumour could be present in the brain without manifesting any symptoms - the patient had been in very good health and had been working up till the day on which her illness began - is remarkable; but it is on record that a man lived for many years with a bullet lodged in the similar cerebral area.

It is quite unusual to find a tumour of such dimensions in a person so young.

Why/

Why the convulsions should have come on and so soon caused death can only be conjecture.

Had there been haemorrhage into or around the tumour resulting convulsions would not have been surprising; but that congestion of the tissues surrounding the tumour, resulting from her alcoholic indulgences, may have caused the convulsions from which the patient was found to be suffering, is the most likely cause that I can suggest.

The literature on this subject is extremely scanty.

CASE II.

Zwiefel records a case which was diagnosed as Eclampsia, and sent to hospital as such. Patient was a multipara and had 88 convulsive attacks - at and after birth - the cause of which was found to be a glioma of the left Cerebral Hemisphere. Albumen in the urine was in this case absent; but with a previous history of some epileptiform convulsions her condition was then regarded as Epilepsy. These attacks certainly gave a hint to the diagnosis.

CASE III.

Snoo records a case in which the patient a multipara - 10 para - had suffered 1½ years from middle ear/

ear trouble.

She was admitted to Hospital in the 9th month of pregnancy with the diagnosis of Eclampsia. Her face was emaciated - there was some oedema of the feet; slight trace of albumen, and middle ear catarrh, also Nystagmus. With the exception of convulsions there were no symptoms present from which cerebral complications could be concluded. There was a spontaneous delivery and slight convulsions during the puerperium. On the eighth day a radical operation was performed and a Cholesteatoma removed from the left Cerebral hemisphere. But the condition remained unchanged. Two days later, the evacuation of a large cerebral abscess was followed by rapid improvement.

CASE IV.

Carl von Hecker records the case of a patient aged 46 - a primipara - who was admitted to hospital in an unconscious condition with the diagnosis of Eclampsia. Half an hour after admission she had a convulsion. The urine showed no trace of albumen. Eleven hours after admission she had five convulsions with very deep coma, and there was noticed left sided facial paralysis. The child was found to be dead - and the os was sufficiently dilated to admit of the child being delivered by forecps. Patient showed

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at first some signs of return to consciousness with a very slow pulse. Delirium was the next feature of the case with a very high temperature. The patient lingered on with a continuing high temperature, and died on the fourth day after delivery. The temperature on that day in the vagina being 106·8^{0} F.

The Autopsy revealed Gelatinous Sarcoma the size of a hen's egg - with some haemorrhage around it in the right Cerebral hemisphere. The tumour arises from the grey matter and lies over the Sylvian Fissure. It showed some fluctuation.

Let us consider these cases just recorded.

Three of them were multiparae and one a primipara.

In Zwiefel's, Snoo's and in Hecker's there was given a slender hint from which the diagnosis could have possibly been made. Zwiefel's case had the history of Epileptiform attacks, so pronounced were they that the patient was diagnosed as an Epileptic.

Snoo's case had middle ear disease for 1½ years and there was also Nystagmus; these two facts pointed to some cerebral lesion at least they were unusual in a case of true Eclampsia and as such it was diagnosed.

Hecker's case had facial paralysis which was the only indication of any palsy; but there was the very slow pulse, which was a significant sign.

In the case under my care there was a continued extensor/

extensor response of the left great toe - which will be held by some as a guide to some nervous lesion - but eminent Neurologists hold that Babinski's sign does not indicate a lesion of the Central Nervous System, for such conditions as extreme cold will cause the response to be made.

In three of the cases there was no albumen in the urine, while in the case recorded by Snoo there was only a slight trace. This is quite explained by the continued temperature and in no way did the albumen present resemble the dense cloud of a typical Eclamptic urine. There was no oedema in any of the cases.

The blood pressure is not recorded in any of the other cases, due to the fact that Sphygmomanometric readings had not come to be regarded as aids to the Clinician, but in my case it was only 140 m.m. of Hg. a much too low reading for true Eclampsia.

The convulsions were in my case atypical in character. In all the cases the convulsions were too numerous for true Eclampsia. In the case recorded by Zwiefel the patient had 88 attacks. Hecker's case had nine in as many hours; and in Snoo's case also they were far too numerous. In my case the convulsions lasted two days and two nights. I cannot give any indication of how many seizures the patient had - there being no record kept before she was admitted to hospital, but the relatives as well as the district nurse/

nurse assert that they were fairly frequent; and this is substantiated by the fact that on the day of admission between 11 a.m. and 12.15 p.m. the patient had six convulsions, and between 12.15 p.m. and 3 p.m. seven convulsions.

Ahlfeld states "An excessive number of Eclamptic attacks from the onset makes the diagnosis of pure Eclampsia extremely doubtful".

Englemann records a case where the patient had 30 attacks before parturition, and 155 after the birth of the child, with recovery.

Jardine reports a case where there were 199 attacks within three days; yet the patient recovered.

Cato records a case of 368 convulsions in one day.

Rissmann put on record a case with 857 attacks, but adds "These attacks should not be regarded as Eclamptic but as Hysterical Manifestations".

It is quite clear that had the convulsions been of the usual severe type associated with Eclampsia death would have supervened long before the total of 155 had been reached.

The absence of albumen, the convulsions atypical numerically as well as in character, the absence of oedema, the low blood pressure, the absence of the livid congested look of the woman were all points in my case that made the diagnosis of Eclampsia doubtful from the first.

CASES OF MENINGITIS.

The next set of cases which I venture to bring forward are cases of Meningitis, simulating Eclampsia. There presented itself at the Royal Maternity Hospital an excellent case of this kind which later proved to be Tuberculous Meningitis.

CASE I.

Case of Tuberculous Meningitis.

Name J. P.

Primipara.

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Admitted December 10th, 1912, 1-30 p.m.

History.

Patient was illegitimately pregnant, and had been at home for a few weeks. She had been doing ordinary house work, as her mother was an outdoor worker.

On December 10th, 1912, before going to work her mother asked patient if she was quite well and a reply in the affirmative was given. But it appears that labour started about 11 a.m. The nurses were summoned and arrived at 11-30 a.m. They found the child born and the placenta expelled. About 12-10 p.m., while the nurses were still in the house patient was seized with a severe convulsion, and a second still more severe/

severe at 1-20 p.m., while yet a third at 1-35 p.m. The case was regarded as one of Eclampsia and sent to the Maternity Hospital, where it was admitted at 2 p.m.

Previous History.

Patient had always been a healthy girl and during her pregnancy had been very well, but being ashamed of her condition was reticent.

State on Admission.

Patient is a fairly well developed girl. Evidently quite recently delivered. She is comatose, but breathing easily if a little quickly. There is no respiratory stertor. She is not cyanosed.

Temperature $99^0.4^0$ F. Pulse 90.

Blood Pressure 120 m.m. of Hg.

Respiratory System. Nothing abnormal to be made out.

Circulatory System. Nothing Abnormal to be made out.

Heart sounds closed and clear at all areas.

Urinary System.

The catheter was passed and 20 ounces of urine withdrawn.

Characters.

Specific Gravity 1020.

Acid.

No albumen.

No blood.

No bile.

No sugar.

No acetone.

No casts.

Progress.

At 2-15 p.m. she had a slight convulsion, only mere twitchings of her face and limbs - no stertor, and no cyanosis.

At 3-30 p.m. Another convulsion similar to the one at 2-15.

4-40 p.m. Had another convulsion, more severe than its predecessor. Slight cyanosis noticeable, and very slight sterterous breathing audible.

5-15 p.m. Had another convulsion. Temperature now 104.6° F. and pulse 126. Showing signs of head retraction.

6·5 p.m. Had another convulsion.

6·30 p.m. Another Convulsion. Hypodermic Injection of Morphia grain ½.

6·45 p.m. Another Convulsion just similar to its predecessors.

7·35 p.m. Another convulsion.

8 p.m. Patient had a very severe convulsion with marked Cyanosis and pronounced Sterterous breathing. This was quite the worst convulsion the patient had had since admission. Ether was administered to relax the spasm.

8.10 p.m. The eyes were examined by an Oculist, but the/

the fundus oculi revealed nothing amiss.

9 p.m. Hot intra uterine douche given. Head retraction very well marked; and at 10 p.m. the diagnosis of Meningitis was almost self evident. Convulsions now recurred with greater frequency, and with increased severity leaving the patient in an extremely exhausted condition. Temperature 105·8°.

10·50 p.m. Lumbar puncture performed and 10 c.c. of cerebro spinal fluid drawn off. The fluid was clear, easily obtained and came off at an exceedingly high pressure.

11 p.m. Temperature 106·8°. Pulse 158.

Midnight. Temperature 108°. Pulse 168. Patient very exhausted, sinking.

1 a.m. Temperature 107° Pulse Imperceptible.

1·25 p.m. Patient died.

Cerebro spinal fluid showed considerable number of Leucocytes - (Lymphocytes.)

Post Mortem Report.

Body well developed and well nourished.

Breasts well developed and showing dark areola.

Abdomen still prominent.

Appendix adherent by apex to portion of omentum.

Appendix extremely bulbous.

Large Caseous Mesenteric Glands.

Kidneys show pallor of cortex.

Liver/

Liver shows no obvious change, but is somewhat pale.
Stomach moderately distended.
Left lung shows adhesions at apex, which is puckered.
Right lung shows no adhesions.
Both Lungs show some oedema and congestion at bases.
Heart. Somewhat soft - no other abnormality.
Brain. Some flattening of convolutions and congestion of vessels. No obvious exudate; excess of cerebro spinal fluid which appears clear. On section brain shows some oedema.
Cord. No obvious change.

Summary:-

Old tubercle of left lung and Mesenteric glands.

Congestion and oedema of lungs and brain.

On microscopical examination of the brain well marked Tuberculous Meningitis was discovered, and that condition was found to be the cause of death.

Unlike the rare frequency of cases of cerebral tumour many instances have been recorded of meningitis in pregnancy and in the puerperum and all of them have been diagnosed as Eclampsia. A great many of these cases have had some focus of pus formation - as middle ear disease - but such was absent in my case.

CASE II

The case occurring earliest in pregnancy of which I can find a record is one reported by Wilson of Birmingham./

Birmingham. She was a woman aged 25, admitted to General Hospital, Birmingham, on April 5th, 1900, with diagnosis of Eclampsia of pregnancy. She was cold and comatose, Pulse 140, Respirations 38, and temperature 100·7° F. When laid in bed the extremities became warm, but pallor soon turned to cyanosis. Respirations became more and more slow, and an hour and a half after admission, patient died without regaining consciousness.

Rapid examination made during her short stay in Hospital showed that she was a multipara pregnant 6 to 7 months:

Heart and Lungs.without abnormal physical signs.

Urine withdrawn by a catheter was

Acid.

Specific Gravity 1032.

Trace albumen and sugar.

No casts.

No Blood.

Her Aunt gave the history that patient was pregnant for the eighth time. Had been very well up till March 31st. Some days before admission she began to complain of violent pain in her head and of general malaise. Headache increased up to April 4th. Next day (April 5th) she was found unconscious in bed. Through a series of generalised convulsions she had involuntary emission of urine.

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A medical man was called, found some albumen in the urine and diagnosed Eclampsia.

At the Autopsy, purulent meningitis was found. No lesion of other organs. The whole of the outer surface of brain was covered with a dense layer of yellowish creamy pus, which extended from the convexity to the base and to the cerebellum. Grey and white substance not altered. Ventricles not distended, no tuberculosis, no pneumonia, no aural lesion, no septic focus. Pneumocci found in purulent exudate of the meninges.

CASE III.

Budin reports a case of Meningitis which occurred at the eighth month of pregnancy.

Woman pregnant about eighth month - admitted to Charti Hospital, Paris, December 8th, 1887.
High Temperature. 103·6^{0}. Pulse 52 Delirious.
Diagnosis made was Eclampsia.
Next day temperature 105·8^{0}. Pulse 160. Marked Delirium. Agitation extreme, necessitating a straight jacket. Mr Budin now diagnosed Meningo encephalitis.- Premature accouchement had been considered but not performed, as patient was so restless.
December 10th, child born. Normal Delivery. Patient died six hours later.
Autopsy./

Autopsy. Acute meningo encephalitis without any trace of tubercle.

CASE IV.

Valentin records a case of Purulent Meningitis in a woman aged 39. 9 para - admitted Maternity Hospital, Nantes with the diagnosis of Eclampsia. Much prostrated and delirious and had had some convulsions. Temperature 104.2°. Typhoid fever diagnosed and transferred to medical wards.

It was ascertained she had severe headache: and slight albuminuria - no oedema - child living. She was venesected. Labour pains began, cervix incised, forceps applied, living child born.

Three days later typhoid stage more accentuated; less delirium: at 4 p.m. she relapsed into profound coma and died in two hours.

Autopsy. No visceral lesions.

Upon opening the cranium intense congestion of whole of meninges. At the base of the right and left hemisphere in the neighbourhood of the fissure of Rolando and beneath the arachnoid, foci of very thickened yellowish pus were found. This pus is found in a thinner layer covering over the whole cerebellum . The lateral ventricles contained a sero purulent exudate.

CASE V.

Beir and Dubrisay report a case.

A primipara - 30 - pregnant and at term.
A few days prior to admission she had a cough and a little headache, and pain in the left ear. This gave no anxiety. No pulmonary symptoms - child living - head engaged - First seen January 13th.
January 15th and 16th a certain quantity of pus escaped from the left ear - discharge stopped spontaneously on January 16th. Shortly after patient experienced severe pain in the head and at night vomited food and somewhat bilious matter in abundance.
From 17th to 18th January she was somewhat better.
On 19th pain in head recommenced and patient was in a state of constant stupor from which she could with difficulty be roused. At this point it was seen she was in labour although she did not complain of painful uterine contractions. At 8 p.m. she was sent to hospital and could without much difficulty answer any questions. Put to bed ½ hour after admission she was spontaneously confined. Temperature was at time of accouchement 99·4° F. Patient continued drowsy about one hour after her accouchement and towards 10 p.m. she began to fling herself about in bed. So violent was she, that it took three persons to hold her/

her in bed. She could not now reply to any questions put to her. At 11 p.m. after morphia she passed into a sound sleep but at 1 a.m. she died very suddenly.

Autopsy - No visceral lesion.

On opening cranium nothing abnormal in the meninges, the dura mater or petrous bone. Meninges easily removed, and on raising the brain, a purulent fluid was found at the level of the bulb. The lesions were limited to the base of the brain in its lateral parts. The pia mater was covered with a purulent exudate more abundant at the fissure of Rolando on both sides.

There was creamy pus in the fourth ventricle.

The lateral ventricles were filled with clear pus.-

Pneumococci were found in the pus taken from the ventricles.

CASE VI.

Brindeau reports a case of acute suppurative meningitis at term.

Primipara. Admitted January 4th, 1903 to St Antoine Maternity with the diagnosis of Eclampsia.

Sharp seizure of malaise - marked headache - convulsions, staring eyes and, soon, coma.

Labour in advance, cervix fully dilated.

Lumbar puncture, turbid fluid showing microscopically staphylococci.

Autopsy.

External/

External aspect of brain covered with thick pus.

CASE VII.

Crougat's case of Pneumococcal. Meningitis one day after accouchement.

A woman aged 40, 12 para - accouchement at term without incident. The day following accouchement in the morning she complained of headache. In the afternoon she began to suckle the child; and at three she had a crisis with violent convulsions. She then fell into a profound coma with copious vomiting. Stiffness of the neck was noticed. Pupils were dilated and unequal. Temperature 102.4° F. Death 24 hours later. Diagnosis lay between Eclampsia and Tuberculous Meningitis or Meningeal Haemorrhage and each was discussed in turn.

Autopsy.

Tracts of yellowish pus along the line of the vessels at the base of the brain. No Tubercle. Cultures showed pure pneumonococci.

CASE VIII.

Brindeau reports a case of Suppurative Meningitis, three days after accouchement.

Woman multipara - Delivery December 9th 1909.

On 12th December sudden loss of consciousness, extreme agitation./

agitation. Taken to St Antonie. Temperature 98.4° F. Upper limbs in state of elbow flexion, contractions of the muscles of the nape of the neck. Pupils dilated. December 12th Temperature 101.2° F. Coma almost permanent. Lumbar puncture yielding purulent fluid, slightly icteric, under microscope show numerous leucocytes and diplococci - streptococci. No autopsy. Eclampsia was first thought of; but the correct diagnosis was made after lumbar puncture.

CASE IX.

Klein records a case which occurred resembling in its commencing stages a case of true Eclampsia in almost all its symptoms but in its further course it was diagnosed as a case of Cerebro Spinal Meningitis. Lumbar puncture was performed and 50 cc of turbid fluid were withdrawn - the fluid spurted out under high pressure and microscopical examination showed the presence of Staphylococcus pyogenes in pure culture. In this instance the patient recovered.

CASE X.

Meyer Wertz reports a case of purulent meningitis that was sent to Hospital under his care as a case of Eclampsia simply because she was a pregnant woman and had developed convulsions.

Meningitis/

Meningitis can simulate Eclampsia in pregnancy, labour or in the puerpurium. Wilson 's case was at the sixth month of pregnancy. Budin's case at the eighth month. Arsoc, and Lafond, and Brindeau each record a case at the ninth month. Genairon and Favre record a case during labour; while my own case occurred a few hours after delivery; Crougat describes a case one day after delivery, and Brindeau, a case three days after delivery.

In all the cases I have brought forward the patients were sent to Hospital with the diagnosis of Eclampsia. All of them had convulsions but none of them the other factor, albumen.

With the exception of two all were multipara ranging up to 12 para - a fact in itself not in accordance with the ordinary history of Eclampsia.
Albumen was either absent from the urine or in a few cases present in such minute quantity that the diagnosis of Eclampsia could not for a moment be entertained.
The quantity of urine obtained from the bladder is allimportant. Twenty ounces withdrawn in my case is all against the diminution as recorded in Eclampsia.
In several cases a clue was given by the fact that the patient had been known to have suffered from middle ear disease, or from a cough with rise of temperature.
Such/

Such history combined with convulsions but no albuminuria must always suggest that the attacks are meningeal and not Eclamptic in origin.
The blood pressure in my case only reached 120 m.m. of Hg. which is far below the usual Eclamptic figure. The number, the rapid recurrence; and the slight character of the convulsions made the diagnosis very doubtful in my case.
Lumbar puncture revealed the diagnosis in several cases, whilst in others - my own for example - the result of lumbar puncture together with the clinical facts and factors substantiated the diagnosis of meningitis.
Lumbar puncture should never be ignored or forgotten in such cases as a means of differential diagnosis.
In my case it was so used - the force with which the fluid came off, the ease with which it came, and its character proved the presence of meningitis.
In Budin's case where no autopsy was granted, lumbar puncture and examination of the fluid enabled the diagnosis of pneumococcal meningitis to be made.
Microscopical examination of the fluid reveals - with the exception of the Tubercle Bacillus - the causal agent of the meningitis, but the presence of Lymphocytes in large numbers suggests Tuberculous Meningitis. A bacteriological examination of the fluid should in all cases be made where the diagnosis is, in any way doubtful.

Examination of the eyes revealed nothing in my case/

case, whilst in Eclampsia Albuminuric Retinitis is to be seen on ophthalmoscopic examination, which should in all cases be carried out.

Many cases of pneumococcal and streptococcal meningitis have been recorded but with the exception of my own, I have been unable to find the record of another case of tuberculous meningitis, although Crougat discussed the possibility of such a condition being present in the differential diagnosis of the case he reported.

HYSTERIA.

Case of Hysteria.

There was sent into the Royal Maternity Hospital a woman aged 40, in labour, with the diagnosis of Eclampsia; and with the history that she had had several Eclamptic Convulsions.

State on Admission.

A fairly well developed healthy looking woman, 10 para - full term - and in labour.
Labour had begun and had been in progress about 8 hours. She had been quite well during all her pregnancy - had no headache, no oedema, and no urinary symptoms.

At first the pains were slight, but after 6 hours they increased in severity, so much so, that the patient could not bear them, and was seized with "convulsions". The "convulsions" consisted of jerkings of the arms and legs and great agitation, followed by apparent loss of consciousness.

The doctor therefore made the diagnosis of Eclampsia, and sent the patient to Hospital. Preparatory to being removed to a cab she was chloroformed. Narcosis was to some extent kept up by a district nurse in the closed conveyance, who asserted that despite her efforts at anaesthetising, the patient continued to have convulsions en route to Hospital.

When/

When she arrived at the Maternity Hospital she was dazed as the result of chloroform inhalation.

She had not the look of an Eclamptic. There was no cyanosis, on the contrary she was rather pale.

Her urine was straightway examined.

A catheter was passed and 30 ounces of urine were withdrawn.

Characters:-

Clear.

Acid.

Specific Gravity 1020.

No albumen.

No blood.

No Bile.

No sugar.

No acetone.

No casts.

The blood pressure was 118 m.m. of Hg.

On abdominal examination the head was found to be engaged and the back of the child lying to the right. Vaginal examination showed os admitting three fingers. Multiparous os. Vertex presenting. ROA; Membranes unruptured.

An hour after admission patient had a convulsion which coincided with a uterine contraction. She waved her arms and jerked her legs about and was very excited and/

and restless. Then followed some jerkings of her arms, but no facial twitchings. This was succeeded by a period of seeming unconsciousness, during which, however, the conjunctival reflex was active. Sterterous breathing and cyanosis were absent.

Patient's pains were few and infrequent. With a few words of encouragement she was put in to a comfortable bed and before long was fast asleep. After a lapse of 12 hours labour pains began; but without convulsions. She made little progress and had eventually to be delivered by forceps.

This case shows how a practitioner could be easily deceived by what might, at first glance appear to be convulsions; but careful investigation would soon reveal the real type of ailment, and show the many points of difference between it and true Eclampsia.

1. She was a multipara.
2. 30 ounces of urine in the bladder.
3. No albumen in the urine.
4. Absence of oedema.
5. Atypical convulsion.
6. Presence of conjunctival reflex.
7. Absence of pathological Blood pressure.

New surroundings, a few kind encouraging words, and a comfortable bed drove away the convulsions and induced/

induced sound sleep; and with it increased strength and vigour.

Commandeur records a case where a woman in the belief that if she feigned convulsions, would have abortion.induced. She was illegitimately pregnant, feigned a convulsion and was sent to Hospital as a case of Eclampsia.

The character of the seizure was noted not to be in accordance with a typical Eclamptic convulsion; so the case was closely observed. The patient got tired being in hospital when nothing was being done, and left the institution.

The severity of labour pains has been recorded as causing convulsions in nervous frightened women, and even in some cases death is said to have resulted.

As a rule, however, such convulsions are, as in the case which I have recorded, imitations. Consciousness is never altogether abolished, as is evidenced by the presence of the conjunctival reflex. The paralytic stage is absent; at the most the attacks are followed by a sense of undue muscular relaxation, or of fatigue, and in general the whole aspect of the case is in harmony with the nature of the attack and not likely to cause the practitioner much anxiety.

Hysteria, therefore, is unlike some of the other convulsions resembling Eclampsia, in as much as the diagnosis is comparatively easy.

Epilepsy/

Epilepsy.

In considering diseases where convulsions play an important part, Epilepsy is the first that occurs to one's mind, as Convulsions and Epilepsy are indissolubly bound together. The diagnosis, however, between Eclampsia and Epilepsy should at this date not present much difficulty.

The abrupt beginning, the absence of history of such a seizure, the albuminous or even bloody urine, the oedema, the precedent headaches and dimness of vision, the frequent return, the intermediate coma, the rapid course and often fatal termination, all characterize a disease which is essentially distinct etiologically, pathologically, and clinically from Epilepsy and to which it bears only a superficial resemblance in its convulsive phenomena.

Gowers states that a convulsion, due to Epilepsy, occurring during the puerperium is unheard of, and it is very rare, Rissman states, for Epilepsy and Labour to be concurrent, so that the diagnosis really lies between Eclampsia in a pregnant woman and an Epileptic, but as I have shown above, that should present very little difficulty.

ALCOHOLISM.

The comatose state consequent upon alcoholic indulgence should not, one would think, present much difficulty in differential diagnosis from Eclampsia. Yet Spielberg records two cases of Drunkenness which were sent to Hospital with the diagnosis of Eclampsia.

The first case, in which the woman was well advanced in pregnancy and took alcohol to such an extent that her condition was mistaken for Eclampsia.

The second case was that of a parturient woman, who feeling labour pains very severe, took alcohol to such an extent that the resulting excited stage and comatose condition were at first believed to be Eclamptic in origin.

A systematic examination of the urine would have found albumen awanting. Gastric lavage would furnish the aroma characteristic of the beverage, but the odour of alcohol would not itself justify the comatose or excited condition being assumed as alcoholism, for alcohol might have been administered perhaps for epigastric pain, to a person really Eclamptic.

TETANUS.

With the perfection of aseptic technique in Obstetrics, Tetanus, once the bugbear of accoucheurs, has proportionately diminished. Such a condition presented a syndrome of symptoms and signs not unlike those of Eclampsia, and therefore falls to be discussed here.

Tetanus was always post partum and occurred in cases where some obstetrical operation had been performed. Thus Sir James Simpson recorded a case after Version, and Gibbons and Gilfons report a case which developed after the introduction of the hand into the uterine cavity to remove an adherent placenta.

The disease begins in the muscles of the neck and may be attributed to Rheumatism, but the nature of the convulsive attacks soon reveals the disease. The evident contractions begin in the muscles of the limbs, spread through the entire muscular system ending by making the patient assume the opisthotonos position.

A marked and distinguishing feature of a tetanic convulsion - and this adds greatly to the horror of the case - is that the patient's intelligence is in no way dulled, in fact often it is hyper acute.

A typical tetanic convulsion is so unlike anything else that observations on differential diagnosis are superfluous.

REFLEX CAUSES.

Turning now to the rarer cases of Pseudo Eclampsia, the first set of cases is composed of those which are ascribed to Reflex Causes. The fons et origo of this irritation is usually the uterus but in some instances the source is in another organ - the bladder for instance.

La Motte has recorded a case which is most instructive, where a woman seven months pregnant, had most violent convulsions. The abdomen was so large that he could not at first believe that her pregnancy was not at its normal end. Learning that she had passed very little urine for some days and that it escaped drop by drop, he attempted to pass a catheter, but finding resistance, he introduced his finger into the vagina and ascertained that the resistance was caused by the head of the foetus which was pressing upon the neck of the bladder. He states "I gently pressed the head as high as possible, and the moment the neck of the bladder was relieved of the pressure and the urine could have free exit, there escaped such an amount that it was impossible to believe that the bladder could contain such a quantity, or that it could be so greatly dilated without rupture". The simple evacuation of the urine ended the convulsions which/

which before threatened a fatal termination.

Speigelberg records a case occurring during labour. The parturient woman was at full time and a primipara. She was siezed with the first convulsion twelve hours after the somewhat premature discharge of the liquor amnii, the second occurred soon after the arrival of the Medical Attendant. Owing to the head lying low in the pelvis and to its compressing the urethra he was unable to evacuate the greatly distended bladder, but extracted the head with forceps. The accumulated urine gushed forth, and on examination proved to be free from albumen. The child lived. There was no return of the convulsions and the lying in woman rapidly recovered.

A case, less conclusive than those recorded by La Motte and Speigelberg - for other remedies were used before catheterisation was employed - in which convulsions were caused by an accumulation of urine has recently been recorded by an American physician. The woman was attacked with convulsions a few hours after delivery - which had been quite normal - and the diagnosis of Eclampsia was speedily made. Veratrum Viride was first given in heroic doses without benefit; then chloral was twice given, first hypodermically and then per rectum. By some happy thought it was deemed wise to examine the urine - a catheter

was/

was accordingly passed and more than a gallon of urine was drawn off. Straightway the convulsions ceased.

Portal and others have recorded similar cases of convulsions due to reflex causes.

These three cases have been chosen because each occurs in a period when Eclampsia can, and usually does, occur, and they are strong points in my contention that in all cases of convulsions a catheter should immediately be passed and a thorough and systematic examination of the urine made.

Had resort been made straight away to the catheter, the patients in these cases I have recorded, would not have been exposed to the grave risk of exhaustion from repeated convulsions and to the unnecessary risks of heroic doses of drugs like veratrum viride and chloral hydrate being administered, for in each case the convulsions subsided as soon as the viscus was emptied.

The urine in each case also, on examination, was found to be free from albumen.

CEREBRAL HAEMORRHAGE.

Cerebral Haemorrhage is usually associated with Arterio Sclerosis accompanied by and due to albuminuria.

Carver and Fairbairn give a most exhaustive and interesting account of many cases of Cerebral Haemorrhage in pregnancy in the puerperium, and in labour - but all these cases had albuminuria. They were cases therefore of true Eclampsia - not Pseudo Eclampsia.

In the literature I have been able to find only one case of Cerebral Haemorrhage assuming the Pseudo Eclamptic type. It is reported by Brooks in the Lancet, May 1st, 1886. He calls it a case of "Eclampsia" beginning two days before, and continuing six days after labour. The urine was repeatedly examined, and found to contain no albumen. The patient had a right sided hemiplegia coming on slowly after a very long convulsion, and gradually disappearing after a few days. The hemiplegia was thought to be caused by a small haemorrhage in the Internal Capsule of the opposite side of the brain.

As such this case is exceedingly rare, and the only parallel case is one related to me by Dr. J. W. Ballantyne. It was the case of a lady, an elderly primipara, who had convulsions after her labour. The urine had been carefully watched during all the pregnancy/

pregnancy, and never had any albumen been detected. The urine at the time of the convulsion contained no albumen. She had only one convulsion and the diagnosis was somewhat doubtful. Six weeks after her confinement she had another convulsion followed by cerebral haemorrhage, and she died soon after.

In light of present day interpretation of such matters I venture to suggest that the first convulsion was really the result of angiospasm of the Cerebral vessels, and that such a condition also existed in the case recorded by Brooks, which I have quoted above, for it is exceedingly rare to have a hemiplegia due to haemorrhage in the Internal Capsule passing off without a trace being left, while with an angiospasm the gradual passing off of the resulting hemiplegia is what clinical history and observation have led us to expect.

CONVULSIONS FOLLOWING THE ADMINISTRATION OF ERGOT.

Black (Glasgow Medical Journal, 1887) records a most extraordinary case where violent convulsions followed the administration of Ergot to a woman who had shown no signs of albuminuria. There had been rather copious haemorrhage and half an ounce of the liquid extract of Ergot was administered. This was quickly followed by violent convulsions. The case was diagnosed as Eclampsia but the condition was, in my opinion, really due to Ergot poisoning.

Hale White says "A huge dose of Ergot" and half an ounce surely was - the recognised dose being 10 to 30 minims, "may produce a peculiar set of symptoms. Tonic contractions of the various muscles, especially those of the extremities, dimness of vision and loss of hearing, and epileptiform convulsions; this variety of ergotism is usually accompanied by vomiting and diarrhoea; and death, occurring from asphyxia due to spasm and weakness of the respiratory muscles."

CONVULSIONS FOLLOWING POST PARTUM HAEMORRHAGE.

That the convulsive seizures often seen shortly before death can present much difficulty of diagnosis, one would hardly credit, yet Speigelberg records a case where the diagnosis did present difficulty. It was a case of Internal Post Partum Haemorrhage and was mistaken for Eclampsia.

It appears that the patient was normally delivered; the family physician had left the house and the patient was in charge of the midwife. Soon after the doctor's departure the patient began to take convulsions. Speigelberg and the family doctor were both summoned and arrived at the house at the same time. The midwife - Speigelberg says - did not realise the true state of affairs, and the doctor put the convulsion down as Eclamptic.

Such a case hardly needs comment, and only a very casual observer with the signs of Internal Post Partum Haemorrhage staring him in the face could confound such a condition with Eclampsia.

INFECTIVE FEVERS.

Infective fevers are often heralded by convulsive attacks which make the case extremely like Eclampsia. This is well seen in pneumonia. In a case recorded by Polano, where a patient sent to hospital as an Eclamptic was found in reality to be suffering from Pneumonia.

Burchardt records a case in the Hofmiere clinic in which on the fourth day of the puerperium, convulsive attacks manifested themselves in a woman previously in quite normal health. There was a sudden rise of Temperature; Pneumonia was found to be the cause of the attacks.

Fleming records a case of disease happily each year becoming less prevalent.

Patient a multipara was delirious. Temperature 101.4°F. Uterine contractions were accompanied by maniacal excitement and convulsive attacks.

The history was that the woman had been quite well and quite conscious till the evening previous, but during the day when she was examined had complained of slight malaise. The breath was offensive and the skin exhaled a very offensive odour. Over the chest and later over the back petechial spots were seen.

Temperature/

Temperature after delivery was 103.6°F. and the patient was removed to the fever hospital where the diagnosis of Typhus fever was confirmed. Several other cases of Typhus fever were also admitted to the hospital from the house where she had lived.

The typical short cough, the rise of temperature, the rusty expectoration along with the physical signs obtained by stethoscopic examination of the chest would reveal the diagnosis. While in the case of Typhus fever the characteristic odour exhaled from the skin at once arouses suspicion which is confirmed when the "Mulberry Spots" pathognomonic of the disease appear.

ACUTE YELLOW ATROPHY OF THE LIVER.

I cannot find in the literature any case of Acute Yellow Atrophy of the Liver actually diagnosed as Eclampsia, but Whitridge Williams says that the diseases are apt to be confounded, so I have thought it advisable to point out the essentials in diagnosis. Case reported by Fredrichs.

P. N. Age 24. Married. 7½ months pregnant. Admitted All Saints Hospital, January 21st.

History.

Patient's friends state that she had not been in her usual good health for two days previously. She had complained of headache and some epigastric pain, and increasing obstinate constipation. She had become suddenly delirious and had to be held down in bed. This culminated in a convulsion.

She was admitted in a deeply comatose condition.

Pulse 80.

Temperature 98.4° and

Respirations 20.

Urine was examined.

Report. Clear, acid, specific gravity 1018.5. No albumen, no bile.

Slight icteric tinge on conjunctivae but this did not extend over the body.

Respiratory/

Respiratory System. Normal.

Circulatory System. Normal.

Liver. Dulness markedly diminished in the axillary line, it only amounted to 1¼ inches.

During the night of 21st and 22nd January, she had several convulsions, and the urine was again examined and showed:-

Specific Gravity 1024. No Albumen. Leucin and Tyrosin crystals in great abundance. Bile.

Patient was suddenly delivered of a seven months foetus, dead, followed by sharp haemorrhage. Patient gradually became more deeply comatose, and died January 23rd.

Post Mortem revealed:-

"Liver lay collapsed against the posterior wall of the abdominal cavity. It was dry and soft. Its capsule puckered and opaque and its margin sharp. The dimensions of the organ were diminished in every direction and particularly in thickness.

The weight of the liver was 1.807 lbs (.82 Kilogrammes) as compared with 4.409 lbs (2 Kilogrammes) the weight of the liver of a normal healthy woman."

The diminution of the liver dulness, the presence of crystals of Leucin and Tyrosin in the urine, and the absence of albumen are the essential points in the differential diagnosis.

CONCLUSIONS.

All convulsive attacks in a pregnant, parturient, or puerperial woman are not necessarily Eclamptic.

I have endeavoured by illustrative cases to show that a clinical picture - which I have ventured to call Pseudo Eclampsia - similar to Eclampsia has been observed in:-

1. Cerebral Conditions.
   - (a) Cerebral Tumours.
   - (b) Cerebral Haemorrhage.
2. Meningitis.
3. Hysteria.
4. Epilepsy.
5. Acute Febrile Diseases.
6. Reflex Causes.
7. Acute Yellow Atrophy of Liver.
9. Post Partum Haemorrhage.
10. Convulsions following administration of Ergot.

All the cases were at one period or another regarded as Eclampsia, and only at the Autopsy was the true lesion found.

In many the urine had never been examined, thereby the great factor, Albumen, was not proved to be present, for Conculsions along with Albuminuria go hand in hand to make the condition Eclampsia - but convulsions themselves/

themselves do not prove the condition to be one of Eclampsia.

Great stress should therefore be laid on the examination of the urine and the following points carefully noted:-

1. The amount obtained by the catheter.
2. The Specific Gravity.
3. Presence of Albumen.
4. Presence of Blood.
5. Presence of Casts.
6. Presence of Acetone.
7. Quantitative Estimation of Urea.

The Blood Pressure should be carefully observed.

Lumbar Puncture should be performed in each case.

Histological and Bacteriological examination of the cerebro spinal fluid thus obtained should be made.

Opthalmoscopic examination should not be neglected.

The Parity of the Patient should be considered.

Character and number of convulsions noticed.

In the event of the case proving fatal a post mortem examination, if possible, should in every case be obtained.

If all these Clinical Methods were systematically used in a suspicious case of convulsions in a pregnant, parturient, or a puerperal woman, many cases which are perfunctorily/

perfunctorily classified as Eclampsia, could be accurately placed under the head of one or other of the diseases which I have grouped together and called Pseudo Eclampsia.

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